A group of asthmatics reported daily symptoms for over seven months while air pollution and weather parameters were monitored. Significant correlations were found between attack rate and pollution levels commonly found in large cities.

Asthma and Air Pollution from a Coal-Fueled Power Plant

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Introduction

Earlier studies of the relationship between air pollution and acute asthmatic episodes have generally been of three types. In the first type, specific allergenic pollutants (e.g., castor bean dust or grain dust) were clearly established as asthmatogenic on the basis of patients' history, skin tests, and re-exposure to the pollutant under control conditions.[1-3] In the second, associations were found between air pollution *in general* and asthma symptoms recorded in diaries, in emergency room, clinic or physicians' records, or during interviews.[4-9] In the third type, the clustering of asthma episodes and their association with low wind speed indicated that an airborne substance was involved, but standard pollution measurements showed no correlation with attack rates.[10-14]

In none of the studies involving the usual ubiquitous pollutants could researchers specify the harmful agent or agents, or find those levels of pollution at which health effects first become marked. Nor could they generally disentangle the effects of air pollution and weather.

The present study was designed to obtain such information. For 7 months daily symptom reports were kept by a panel of asthmatics who were intermittently exposed to high levels of pollution because their homes were within 1/2 mile of a coal-fueled power plant. At the time of the study, the plant had low stacks, no abatement devices, and used high-ash, high-sulfur coal as fuel, and therefore emitted substantial quantities of particulates, sulfur dioxide and oxides of nitrogen. Our purpose was to quantitate the relationship between short-term relatively high-dose pollution exposure and frequency of asthma episodes.

Methods

Population Selection

New Cumberland, West Virginia was chosen for the present study because air quality measurements showed wide day-to-day fluctuation of pollution levels, and because its small area (1 mile by 1/2 mile) made intensive monitoring of pollution levels relatively simple. Subjects were located by telephone survey of all households in the small town (population 2,100). All residents claiming respiratory symptoms were screened by a staff physician (A.C.). Criteria for inclusion in the study were two:

1. History of intermittent episodes of respiratory distress, generally unaccompanied by fever or increased sputum production, in which wheezing was the predominant symptom.
2. Experience of three or more such episodes in the preceding 12 months.

Most subjects had been told by their physician that they had "asthma," "allergenic asthma," or "asthmatic bronchitis." Each subject was asked if he knew of neighbors with respiratory symptoms; this enabled us to find several subjects among the few local families without telephones.

Forty-three suitable subjects were identified. Of these, 14 were lost to the study, because of refusal to be interviewed (1), protracted hospitalization or travel (2), or repeated failure to return diary forms (11). Of the remaining 29 subjects, 9 reported no attacks or one attack during the study. All analyses presented are for the data of the 20 subjects who reported more than one attack.

A questionnaire administered to the 42 initial subjects inquired into age, sex, race, education, rent paid, duration and severity of asthma, medicine usually taken, symptoms during and between attacks and history of smoking and allergy. The distribution of these characteristics among the 14 study dropouts was not significantly different from that of the final participants. The group of subjects excluded because their attacks were too infrequent was younger, had fewer smokers, was slightly better educated and paid slightly more rent than the final study group.

Monitoring of Pollution and Weather

Three pollution monitoring stations were established at sites picked to give a representative sampling of population exposure. At each station, continuous measurements made included total suspended particulates (High Volume Sampler –24 hour samples),[17] sulfur dioxide (Coulometric method),[18] soiling index (AISI tape sampler–2 hour tape samples),[19] suspended sulfates (24-hour samples,[20]) and suspended nitrates (24-hour samples[20]).

At one weather station in the center of town, and one on a nearby ridge, meteorologists took continuous hourly measurements of temperature, wind speed and direction, humidity and barometric pressure.

Diary Reports

Each subject received a diary form (Figure 7) and return envelope weekly. The diary form asked day of week, time of day, duration, severity, and place of onset for each episode. Three to five days after the end of each weekly reporting period, nonrespondents were phoned and reminded to submit the missing report. Each week approximately 15 per cent of the study population had to be telephoned.

Physician Visits

A staff physician made several week-long visits to New Cumberland. During these visits, subjects were asked to contact the physician each time they had an attack. The doctor then visited each one to confirm the occurrence of an asthma episode.

Analysis of Data

Methods of analysis included a stepwise multiple regression procedure with an analysis of the correlation coefficients, for log-transformed as well as for untransformed data. The association between attack rate and each pollutant was examined within three temperature ranges, as was the association of temperature with attack rate within three pollution ranges. Regressions were also calculated for each pollutant on the temperature-adjusted attack rate. Best-fit lines were plotted for each pollutant against attack rate and against temperature adjusted attack rate.

Results

The Population

Table 1 gives the population's age, sex, duration and severity of asthma, allergy and smoking history, and educational level. The final study group contained primarily adults (80 per cent), with at least a high school education. About half the group were male, half were smokers, half had a history of allergy, and all were Caucasian.

Table 1—Characteristics of the Study Population

Variable	Category	
Age	**16 or less**	**Over 16**
	4	16
Sex	**Male**	**Female**
	9	11
Duration of Asthma	**10 years or less**	**More than 10 years**
	9	11
Frequency of Episodes	**12/year or less**	**More than 12/year**
	0	20
(Non-asthmatic) Allergic History	**Positive**	**Negative**
	10	10
Smoking History	**Smoker**	**Non-smoker**
	11	9
Educational Level	**Did not graduate HS**	**HS or more**
	7	13

Association of Attack Rates with Pollution or Weather Parameters: Two-Variable Analysis

Averages—Correlation coefficients were calculated between daily averages of each measured pollution or weather parameter and reported daily attack rate, and then between pollution and weather parameters. As the first column of Table 2 reveals, *all* pollutants as well as temperature correlated significantly with attack rate, although temperature, sulfur dioxide, and soiling index showed the greatest correlations. Figure 1 shows scattergrams for attack rate versus each variable that was significantly associated with attack rate. "Best-fit" linear regression lines are superimposed on the scattergrams. We attempted to fit threshold functions, i.e., S-shaped curves to the data. For each variable, however, the best fit S shaped curve did not explain more attack rate variation than the best-fit linear-regression line.

Lags—The data were re-analyzed assuming 6, 12, 18 or 24 hour lags between pollution or temperature levels and occurrence of asthma episodes. Temperature and soiling index remained significantly correlated with attack rate with

Figure 1A—Scattergrams With Best-Fit Lines for Attack Rate versus Significant Variables

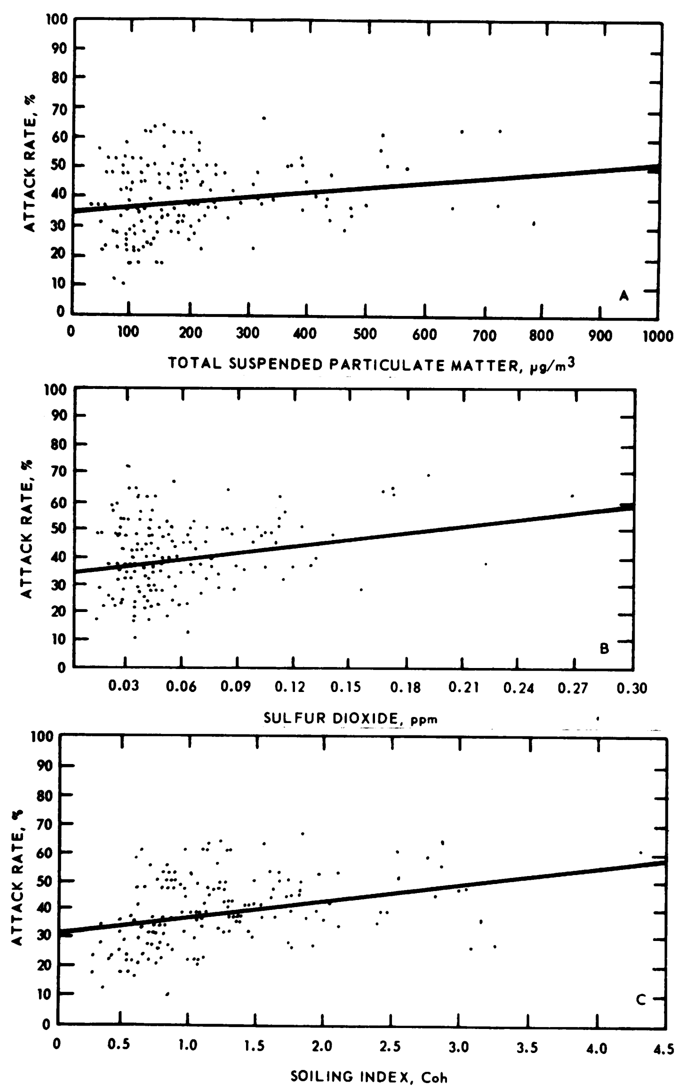
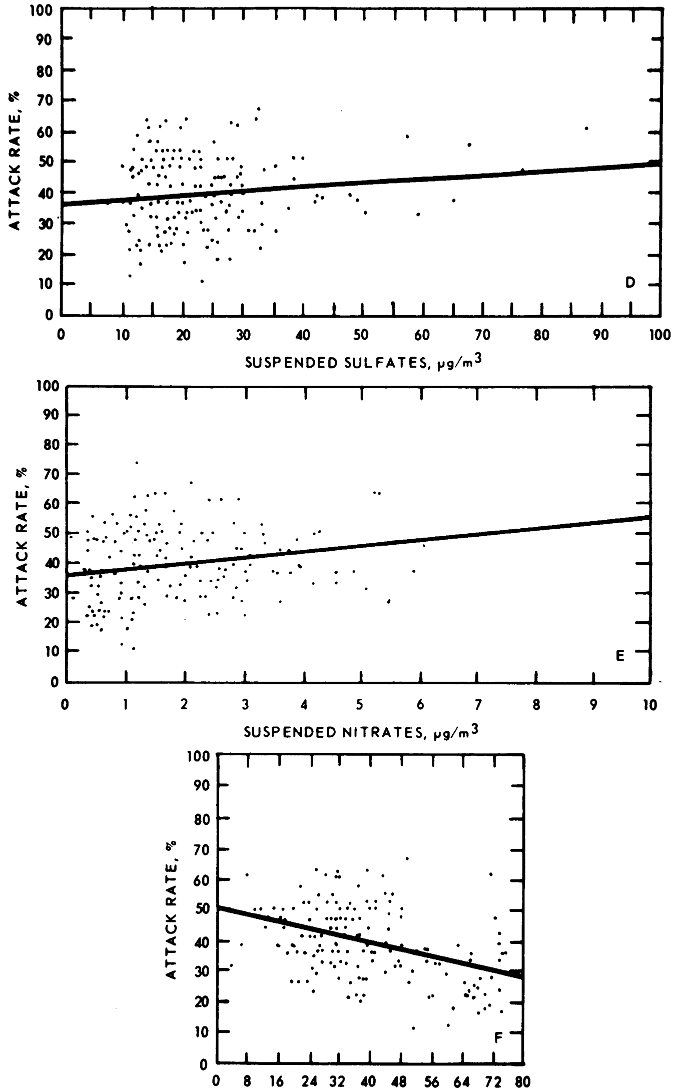


Figure 1B—Scattergrams With Best-Fit Lines for Attack Rate versus Significant variables



any of these lags. The correlation between attack rate and sulfur dioxide level decreased with increasing lag, becoming insignificant at lags of 18 hours or more.

Peaks—Analyses were repeated for all variables characterizing 6-hour temperature rather than by 24-hour averages. Correlations were virtually identical with those found for daily averages.

Too few days occurred with a low 24-hour pollution average but a high 6-hour peak, or with a high 24-hour average temperature but a low 6-hour minimum for us to make a meaningful analysis. The present study therefore could not distinguish the effects of short term (6-hour) high pollution exposure from those of longer term (24-hour), lower dose exposure.

Logs—Analysis of averages, lags and peaks of log-transformed data produced results no different from those described above.

High-low Analysis—Table 3 gives attack rates for high- and for low-pollution days and for days of high and low temperature, humidity, barometric pressure and windspeed. Cut off points demarcating "high" from "low" variable days were arbitrarily chosen so as to roughly equalize the number of days in each category and because these were points at which relatively large increases of attack rate occurred. In addition the table shows relative risks of high pollution, high humidity, and low windspeed, low temperature and low barometric pressure. Relative risk of high pollution varied from 1.03 for suspended sulfates and suspended nitrates to 1.24 for soiling index. The relative risk of low temperature was 1.29. Significant High-low differences in attack rate were found for suspended particulate, soiling index, sulfur dioxide, temperature, humidity and windspeed.

General Temporal Patterns—Temperature, sulfur dioxide and soiling index, the three variables showing highest correlations with attack rate, were tabulated with attack rate by quarter of day, day of week, and month of year.

Pollution levels and attack rate both peaked during the second quarter of the day; over half of all attacks were reported to have begun during this interval. Temperature was lowest between midnight and 6 a.m.

Figures 2 and 3 show average values of temperature, sulfur dioxide, soiling index and attack rate as functions of

Table 2—Correlation Coefficients for Pollution and Weather Parameters, and Attack Rate

	Attack rate	Sulfur dioxide	Soiling index	Temperature	Total Suspended particulates	Suspended nitrates	Suspended sulfates
Attack rate							
Sulfur dioxide	+.320†						
Soiling index	+.387†	+.525†					
Temperature	−.427†	−.205†	−.466*				
Total suspended particulates	+.241†	+.537†	+.419†	−.021			
Suspended nitrates	+.169*	+.316†	+.488†	−.084	+.543†		
Suspended sulfates	+.199†	+.349†	+.480†	+.197*	+.567†	+.361†	
Barometric pressure	+.038	−.055	+.122	−.043	+.156*	+.165*	+.107
Windspeed	+.050	+.072	−.290†	−.124	+.033	−.023	+.267†
Humidity	+.112	−.112	+.223	−.162*	−.168*	−.186*	+.089

* $P<.05$
† $P<.01$

day of week and month of year. Figure 2 shows a "middle of the week" trend for attack rate and pollution, but no trend for temperature. Figure 3 shows average monthly attack rates varying directly with average monthly pollution levels and inversely with temperature; these relationships obtained in *each* season studied. Identical tabulations of the other pollution variables, though not presented here, showed similar characteristics.

As these figures and Table 2 show, each variable significantly correlated with attack rate was correlated with several other variables as well. These simple analyses thus failed to distinguish between primary and secondary associations.

Figure 2—Attack Rate, Temperature, Sulfur Dioxide Concentration, and Soiling Index as Functions of Day of Week

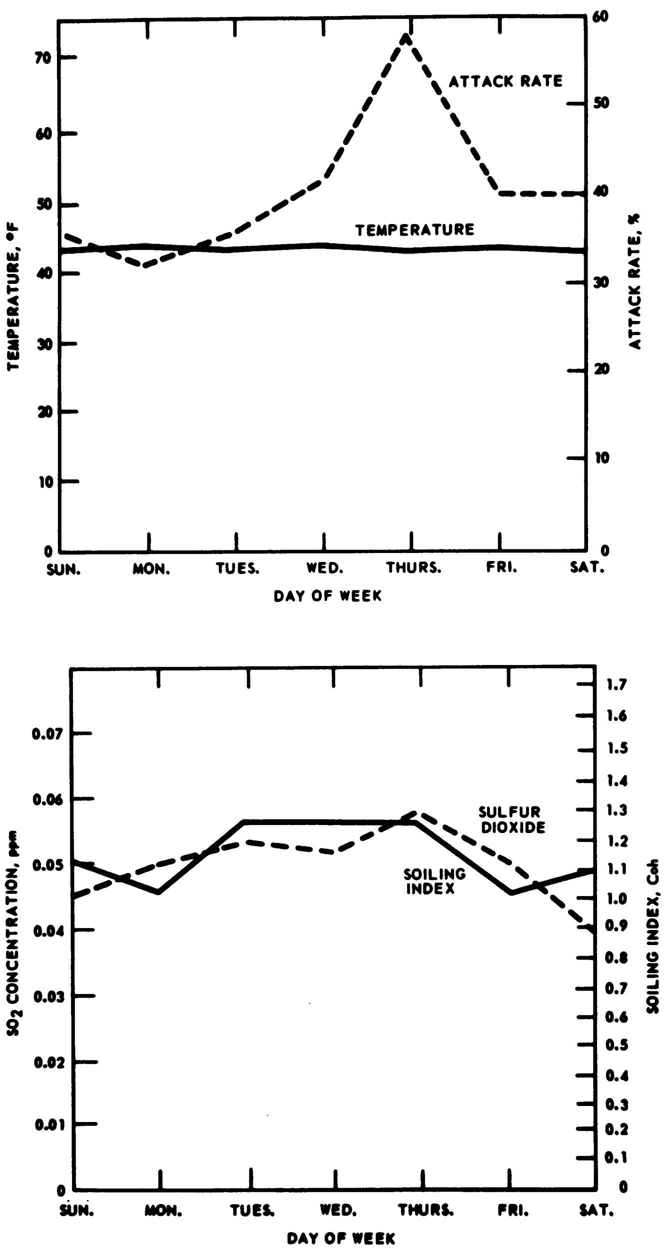
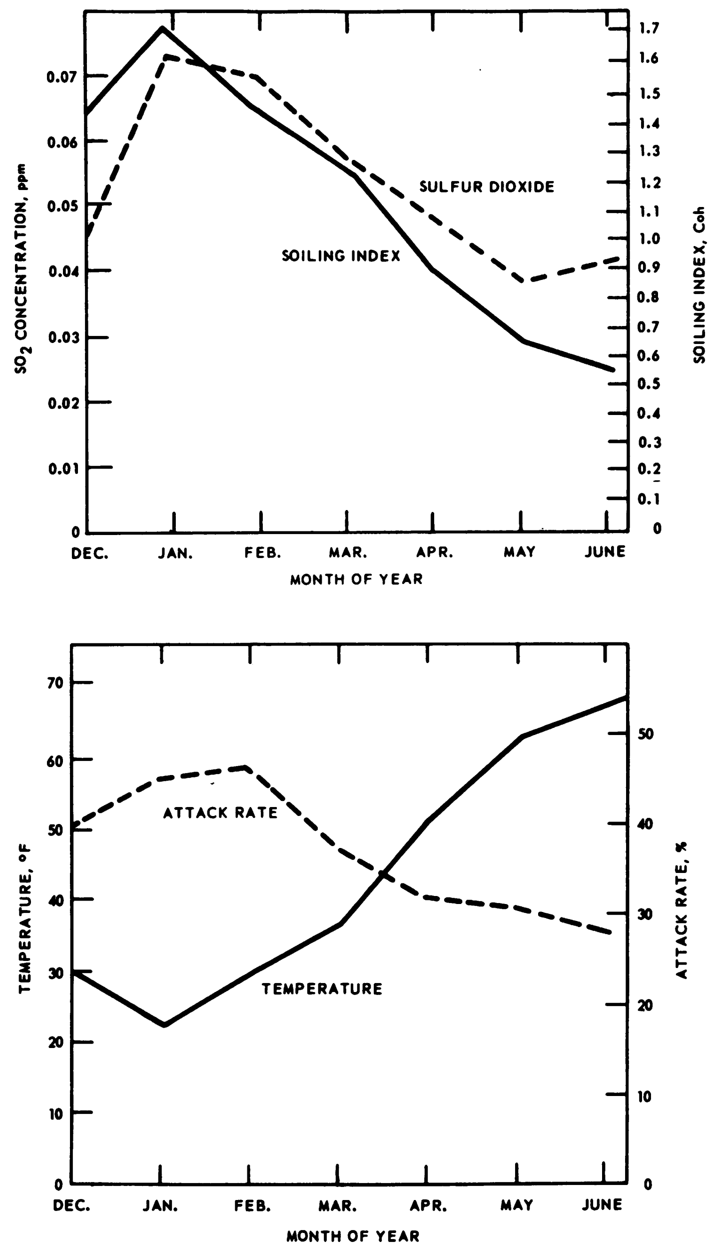


Figure 3—Attack Rate, Temperature, Sulfur Dioxide Concentration, and Soiling Index as Functions of Month of Year



Separation of the Effects of Weather and Pollution Parameters: Multivariate Analysis

Since temperature showed by far the strongest association with attack rate, each variable was examined separately after the effects of temperature were removed in a multiple-regression analysis. Sulfur dioxide, soiling index, total suspended particulates, suspended sulfates and suspended nitrates were each found to explain a significant amount of residual variation in attack rate (Table 4). After temperature and *any one* of these five variables had been taken into account, none of the other four variables explained a significant amount of attack rate variation. These results are confirmed in Figure 4, which shows best-fit lines for

Table 3—Asthma Attack Rate and Pollution and Weather Variables: High-Low Analysis

Variable	Attack rate on "low" variable days (A)	Point demarcating "high" and "low" days	Attack rate on "high" variable days (B)	Relative risk of "high" variable days (B/A) or low Temperature (A/B)	Significance of differences in mean attack rates
Soiling index	0.342	1.0 Coh	0.422	1.24	$P<.01$
Sulfur dioxide	0.364	.07 ppm	0.448	1.23	$P<.01$
Total suspended particulate	0.349	150 $\mu gm/m^3$	0.412	1.19	$P<.01$
Suspended nitrates	0.374	2 $\mu gm/m^3$	0.388	1.03	NS
Suspended sulfates	0.378	20 $\mu gm/m^3$	0.382	1.03	NS
Temperature	0.441	32°F	0.346	1.29	$P<.01$
Barometric pressure	0.380	29.4 inches Hg	0.374	0.98	NS
Windspeed	0.367	4 mph	0.391	1.06	$P<.05$
Humidity	0.362	80%	0.393	1.09	$P<.05$

"temperature-adjusted attack rate" against each pollution variable. On these graphs the difference between observed attack rate and attack rate predicted from temperature data is plotted against pollution level. The best-fit lines are all of significant slope and explain a significant proportion of residual attack rate variation. This series of analyses thus showed independent effects of temperature and of air pollution on attack rate, but could not specify which pollutants were most important.

Figure 4—Temperature-Adjusted Attack Rate versus Pollution Levels: Best-Fit Lines

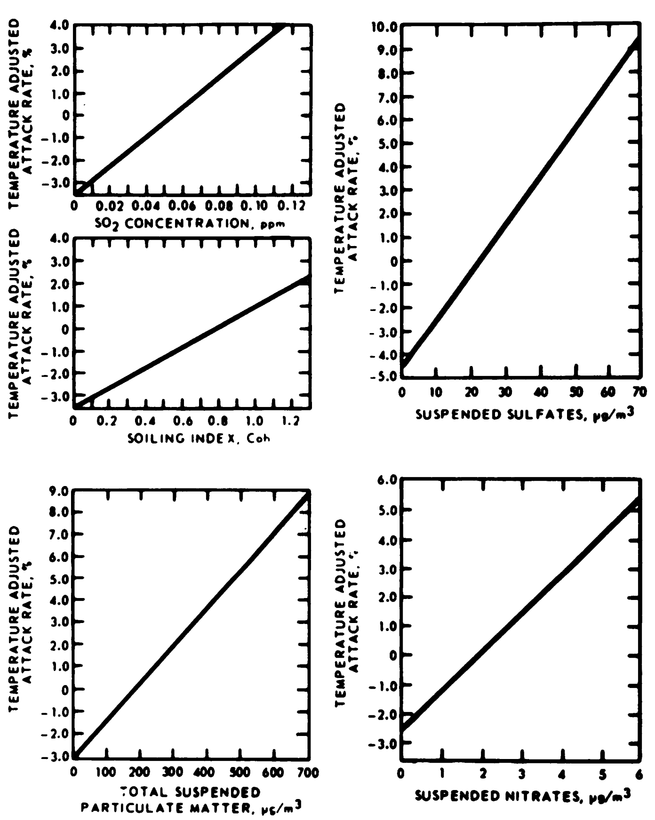


Figure 5 shows best-fit lines for attack rate versus each pollution variable as a function of temperature range. These graphs show that air pollution's effect on asthma is greater at moderate temperatures than at temperatures below 30°F, but that asthma attack rate is greatest at low temperatures.

Figure 6 shows best-fit lines for temperature versus attack rate for three pollution levels and two indicator pollutants, sulfur dioxide and soiling index. These reveal that temperature variation has a greater effect on attack rate at low pollution levels than at high pollution levels.

Physician Visits

The attack rate reported during the several physician visits was not significantly lower than that reported for other weeks matched for season and general pollution level.

Table 4—Residual Variation in Attack Rate Explained by Each Variable After Effects of Temperature are Removed

Factor	Degrees freedom	Sum of squares	% of sum of squares	F-value	P
Total		2.5363	100		
Temp (alone)	1	0.5151	20.31	38.7395	.00005
Temperature with:					
AISI	1	0.0996	3.92	7.8268	.00585
SO_2	1	0.1002	3.95	7.8772	.00570
TSP	1	0.0979	3.86	7.6875	.00630
Nitrates	1	0.0601	2.36	4.6265	.03315
Sulfates	1	0.1391	5.48	11.1641	.00106
Barometric Pressure	1	0.00003	0.00	0.0022	.9627
Windspeed	1	0.0044	0.17	0.3271	.5685
Humidity	1	0.0047	0.18	0.3544	.5526

Figure 5—Attack Rate versus Pollution Levels Within Three Temperature Ranges: Best-Fit Lines

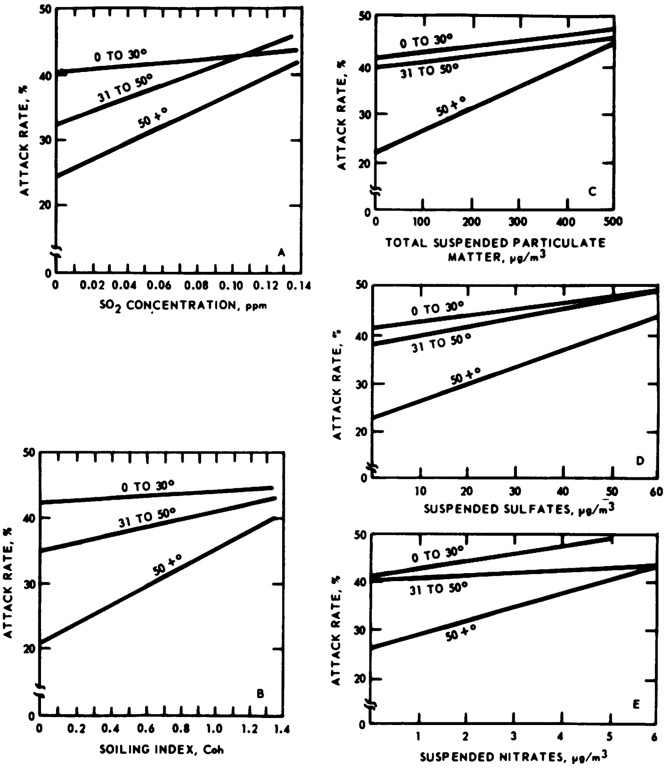
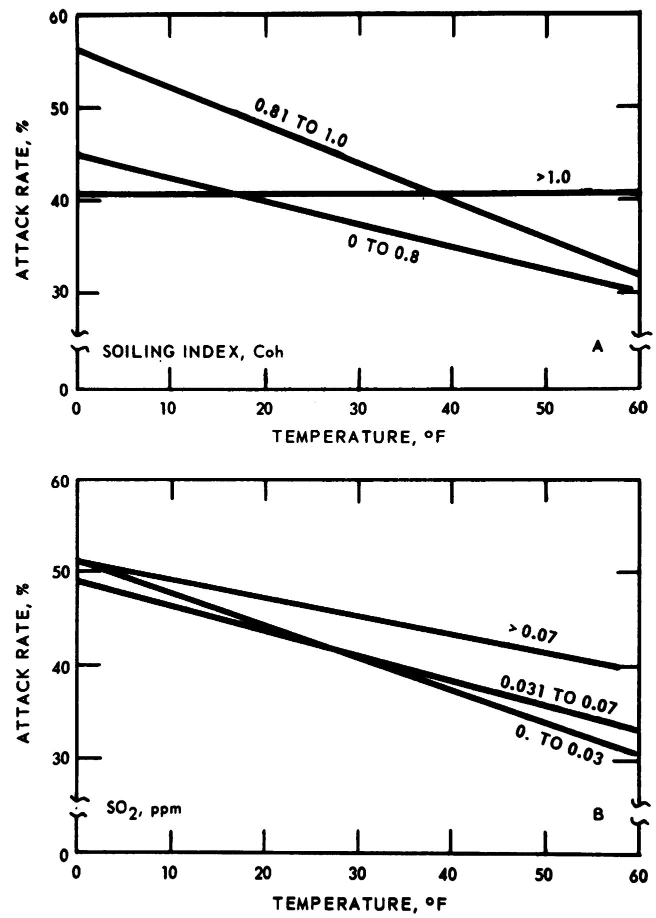


Figure 6—Temperature versus Attack Rate for Each of Three Pollutant Concentration Ranges: Best-Fit Lines



Discussion

We had feared that New Cumberland residents, in their discontent with the local power company, would over-report illness on days when pollution levels were visibly elevated. However, since attack rates reported during the 3 weeks in which a doctor confirmed the presence of each asthma episode were not lower than those reported for preceding or following control weeks, it is unlikely that this was a significant bias.

We attempted to judge severity of attack by inquiry into duration of attack, restriction of activities, dose of medicine taken, and whether or not a doctor visit was made. These proved to be of little value for this population, because little within-person, between-attack variation, occurred. Most subjects rarely saw a doctor, rarely stayed home from work or school because of an attack, took one dose of medicine, and had symptoms for less than 2 hours.

Several aspects of the data deserve further comment. First, pollution levels and attack rates were associated *within* each season. The months of peak attack rate were January and February. These months also had the highest pollution levels and lowest temperature levels, and are characteristically the time when acute respiratory infections are most frequent.

Second, the diurnal pattern for reported attack rate in this study showed the peak rate of symptom onset occurring between 6 a.m. and noon, rather than between midnight and 6 a.m., as reported elsewhere.[6] The reason for this is unclear.

Third, suspended sulfate levels showed the strongest association with attack rate after the effects of temperature were removed. This finding may reflect only the unusual *positive* correlation of suspended sulfates and temperature (all other pollutant variables show negative or insignificant correlations), or may reflect the irritative properties of sulfuric acid mist. Previous studies have shown that sulfuric acid mist is capable of increasing pulmonary resistance in healthy adults,[21] and have shown associations of mortality with suspended sulfate levels.[22]

A single pollutant cannot, on the basis of this data, be singled out as the prime cause of asthma episodes. It is clear, however, that although temperature is strongly associated with attack rate, air pollution concentrations are significantly associated with attack rate, too, even after the effects of temperature have been removed.

Increases in air pollution concentrations were found to have a greater effect on asthmatics when temperatures were moderate than when they were below freezing (Figure 5), although the reported attack rate on days of low temperature was usually high regardless of air pollution concentrations. Similarly, decreases in temperature had a greater effect on attack rate on low pollution days than on high pollution days. On high pollution days attack rates were unusually high, and were not significantly affected by temperature variation. These data seem to imply that there is an upper limit to the ability of environmental factors to bring on asthma episodes, at least in the temperature and pollution ranges studied. Once this limit has been reached, by virtue of low

Figure 7—New Cumberland Asthma Diary

Division of Health Effects Research
National Air Pollution Control Administration
Public Health Service, DHEW

NEW CUMBERLAND ASTHMA DIARY

Date ______________ No. ________ Your Name ______________

		Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
Did you have an asthma or wheezing attack today?	No							
	Yes: 1 attack							
	Yes: more than 1							
At what time did the attack begin?	Midnight to 6 AM							
	6:01 AM to Noon							
	Noon to 6 PM							
	6:01 PM to midnight							
Were you in New Cumberland when the attack began?	No							
	Yes							
How many doses of medicine did you take?	None							
	1 dose							
	2 doses							
	more than 2 doses							
Was the attack bad enough to make you see a doctor?	No							
	Yes							
Was the attack severe enough to keep you from school or work, or from your usual activities?	No							
	Yes							
What do you think brought on the attack?								
How long did the attack last?	Less than 1/2 hour							
	1/2 to 2 hours							
	2 to 4 hours							
	More than 4 hours							

temperature or high pollution levels, further variation in other environmental factors has no effect.

The temperature-independent pollution effects occurred at pollution levels commonly found in our major cities. In New York City, for example, for the months June through November 1969, 14 of 38 representative monitoring stations reported sulfur dioxide averages greater than .07 ppm, and 33 of 38 reported average soiling indices greater than 1.0 Coh.[23] The city-wide average sulfur dioxide level for Chicago exceeded .065 ppm in 1966 and 0.07 ppm in 1967, with corresponsingly high particulate concentrations.[24] Furthermore, as the need for electric power increases, expansion of existing coal-fueled power plants and the construction of new plants appear inevitable. The present data should emphasize the desirability of expanding facilities in sites relatively distant from population centers.

Previous attempts to relate air pollution to asthma have been blocked by several problems. Pollution monitoring has been inadequate because of unsophisticated instrumentation or the impossibility of continuous monitoring.[1-5,7-9,15,16] Procedures for quantifying morbidity have been inaccurate or insensitive.[4,8,9,15] Terminology has been unclear.[8-9] It has not been possible to separate the effects of air pollution from those of temperature, season, barometric pressure or windspeed, or to reasonably estimate dose response relationships between air pollution and health effects.[4-15]

In the present study, weather and pollution were intensively and continuously monitored, a fairly sensitive device was used to measure attack rate, checks on biases were made, and a clear, generally acceptable definition of asthma was used. With these precautions, significant effects of pollution have been found independent of, or in addition to, those of weather. In this study, too, certain desirable information could not be obtained. The effects of the several pollutants could not be separated, since they came from the same source and accumulated under the same inversion conditions. For both pollution and temperature, it was not possible to tell whether 6-hour averages or 24-hour averages were of greater predictive value for asthmatics. The effects of sharply falling barometric pressure could not be evaluated, since no such drops occurred. Finally, since there were no nearby communities without significant pollution exposure, there were no area controls. The conclusions of the present study would be strengthened by finding that a group of asthmatics in a nearby but minimally polluted area did not always show symptoms increases on the same days as the New Cumberland asthmatics.

It is clear that similar studies in other areas and with more subjects would be desirable.

Summary

Twenty asthmatics gave daily symptom reports for over 7 months, while local air pollution and weather param-

eters were intensively monitored. Significant correlations were found between reported attack rate and temperature and between attack rate and pollution levels after the effects of temperature had been removed from the analysis. These temperature-independent air pollution effects occurred at levels of pollution commonly found in large cities, and appeared greater at moderate than at low temperatures.

References

1. Figley, K. D. and Elrod, R. H. Endemic Asthma Due to Castor Bean Dust, JAMA, 90:79-82, 1928.
2. Mendes, E. and Centra, A. B. V. Collective Asthma Simulating an Epidemic Provoked by Castor Bean Dust. J. Allerg. 25:253-259, 1954.
3. Cowan, D. W.; Thompson, H. J.; Paulus, H. J.; and Mielke, P. W. Bronchial Asthma Associated with Air Pollutants from the Grain Industry. J. Air Poll. Contr. Ass. 13:546-552, 1963.
4. Gersh, L. S.; Shubin, E.; Dick, C.; and Schulaner, F. A. A Study on the Epidemiology of Asthma in Children in Philadelphia. A. Allerg. 39:347-357, 1967.
5. Schrenk, H. H.; Heimann, H.; Clayton, G. D.; Gafafer, W. M.; and Wexler, H. Air Pollution in Donora, Pennsylvania: Epidemiology of the Unusual Smog Episode of October, 1948. Public Health Bulletin No. 306, 1949.
6. Schoettlin, C. E. and Landau, E. Air Pollution and Asthmatic Attacks in the Los Angeles Area. Public Health Rep. 76:545-548, 1961.
7. Zeidberg, L. D.; Prindle, R. A.; and Landau, E. The Nashville Air Pollution Study: I. Sulfur Dioxide and Bronchial Asthma: A Preliminary Report. Amer. Rev. Resp. Dis. 84:489-503, 1961.
8. Yoshida, K.; Oshima, H.; and Imai, M. Air Pollution and Asthma in Yokkaichi. Arch. Environ. Hlth. 13:763-768, 1964.
9. Yoshida, K.; Oshima, H.; and Imai, M. Air Pollution in the Yokkaichi Area with Special Regard to the Problem of "Yokkaichi Asthma." Industr. Hlth. 2:87-94, 1964.
10. Lewis, R.; Gilkeson, M. M.; and McCalden, R. O. Air Pollution and New Orleans Asthma, Public Health Rep. 77:947-954, 1962.
11. Weill, H.; Ziskind, I. M.; Dickerson, R.; and Derkes, V. J. Epidemic Asthma in New Orleans. JAMA, 190:811-814, 1964.
12. Weill, H.; Ziskind, M. M.; Derkes, V.; Lewis, R.; Horton, R. J. M.; and McCalden, R. O. Further Observations on New Orleans Asthma. Arch. Environ. Hlth. 8:184-187, 1964.
13. Lewis, R.; Gilkeson, M. M.; and Robison, R. W. Air Pollution and New Orleans Asthma. 2 volumes. New Orleans: Tulane University, 1962.
14. Carroll, R. E. Epidemiology of New Orleans Epidemic Asthma. AJPH, 58:1677-1683, 1968.
15. Sultz, H. A.; Feldman, J. G.; Schlesinger, E. R.; and Mosher, W. E. An Effect of Continued Exposure to Air Pollution on the Incidence of Chronic Childhood Allergic Disease. AJPH, 60: 891-900, 1970.
16. Cowan, D. W. and Paulus, H. J. Relationship of Air Pollution to Allergic Diseases. Final Report under Research Grant AP00090, Division of Air Pollution, Department of Health, Education and Welfare, U. S. Public Health Service, Washington, D.C. 136 pp. 1969.
17. Jutze, G. A. and Foster, K. F. Recommended Standard Method for Atmospheric Sampling of Fine Particulate Matter by Filter-Media High Volume Samplers. JAPCA 17:17, 1967.
18. Beckman Instruction Booklet No. 1636. Beckman Instruments Incorporated, Fullerton, California April 1967.
19. Hemeon, W. C. C.; Hainer, G. E., Jr.; and Ide, H. M. Determination of Haze and Smoke Concentration by Filter Paper Samplers. Air Repair 3:22-28, 1953.
20. Selected Methods for the Measurements of Air Pollutants. U. S. Department of Health, Education and Welfare, National Air Pollution Control Administration, Publication No. 999-AP-11, May, 1955.
21. Sim, V. M. and Prattle, R. E. Effect of Possible Smog Irritants on Human Subjects. JAMA, 165:1908-1913, 1957.
22. Slater, P. B. Atmospheric Sulfate and the Analysis of the Structure of Age-Sex-Disease Specific Mortality Rates. Masters dissertation, Regional Science Department, Univ. of Penn., 1970.
23. Monthly Summaries of Air Pollution Data, New York City Department of Air Resources, Bureau of Technical Services, June 1969-November 1969.
24. Carnow, B. Methodology for Assessing the Relationship of Air Pollutants to Mortality. Presented at the Annual Meeting of the American Public Health Association, Houston, Texas, October 26, 1970.

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